Autophagy Antibodies
Abgent: your partner in autophagy research

Abgent has the most extensive collection of autophagy antibodies. From the hallmark autophagy antibody LC3 to the newest autophagy antibodies such as LAMP and APG1, Abgent offers the most relevant, qualified antibodies for autophagy research. Its expanding collection includes hundreds of antibodies targeting autophagic proteins.

Autophagy is a catabolic trafficking pathway for bulk destruction/turnover of long-lived proteins and organelles via regulated lysosomal degradation. This process has recently been shown to be important in cancer, neurodegenerative diseases, and cardiovascular diseases. The process consists of sequential signaling, sequestration of cytoplasm, formation of a unique double membrane vesicle (autophagosome), targeting of the completed vesicle to the lysosome followed by docking and fusion, and breakdown. Detailed reviews of autophagy and its associated proteins can be found on page 4.
## Autophagy Expansion & Apoptosis

Major proteins associated with autophagy and their interactions during the autophagy process.

The network above is from the Abgent Macro Autophagy wall chart, an overview of the autophagic process. Request a FREE copy at www.abgent.com.

## Autophagy & Disease

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>ACTIVATION OF AUTOPHagy</th>
<th>INACTIVATION OF AUTOPHagy</th>
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<tbody>
<tr>
<td>Cancer</td>
<td>Blocks tumor growth.</td>
<td>Favors tumor growth. Makes cells unable to enter autophagic cell death after exposure to anticancer treatments.</td>
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<tr>
<td>Early stages</td>
<td>Favors survival of cells in low-vascularized tumors. Favors removal of damaged intracellular macromolecules after anticancer treatments.</td>
<td>Prevents survival of cells in low-vascularized tumors. Increases efficiency of anticancer treatments because damaged macromolecules cannot be eliminated.</td>
</tr>
<tr>
<td>Late stages</td>
<td>Favors survival of cells in low-vascularized tumors. Favors removal of damaged intracellular macromolecules after anticancer treatments.</td>
<td>Prevents survival of cells in low-vascularized tumors. Increases efficiency of anticancer treatments because damaged macromolecules cannot be eliminated.</td>
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<tr>
<td>Vacuolar myopathies</td>
<td>Promotes elimination of the cytosolic autophagic vacuoles and protein aggregates.</td>
<td>Results in the accumulation of autophagic vacuoles that weaken skeletal and cardiac muscles.</td>
</tr>
<tr>
<td></td>
<td>If hyperactivated could result in muscle waste.</td>
<td></td>
</tr>
<tr>
<td>Neurodegeneration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early stages</td>
<td>Favors removal of cytosolic protein aggregates.</td>
<td>Increases accumulation of cytosolic protein aggregates.</td>
</tr>
<tr>
<td>Late stages</td>
<td>Destroys irreversibly damaged neurons by autophagic cell death.</td>
<td>Results in accumulation of autophagic vacuoles that alter vesicular trafficking.</td>
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<tr>
<td>Axonal injury</td>
<td>Favors removal of neurotransmitter vesicles and damaged organelles.</td>
<td>Prevents removal of damaged organelles and neurotransmitters vesicles.</td>
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<tr>
<td></td>
<td>Provides energy and membranes for regeneration.</td>
<td>Cytosolic release of neurotransmitters induces apoptosis.</td>
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<tr>
<td>Infectious disease</td>
<td>Contributes to the elimination of bacterial and viral particles.</td>
<td>Offers a survival environment for the bacteria that are able to inhibit autophagosome maturation. Facilitates viral infection.</td>
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</table>

Occurrences that favor progression of the disease are noted in light gray boxes.
Autophagy, a Survival Guide

Two cellular instruction sets regulating survival/extinction at the individual level of shortlived proteins (the ubiquitin pathway) and at the grand level of the cell (apoptosis) have received great attention over the last fifteen years; the emerging details have advanced both fundamental research and therapeutic initiatives. Autophagy, the third leg on this stool, is a catabolic trafficking pathway for bulk destruction/turnover of long-lived proteins and organelles via lysosomal degradation. Newer techniques permitting the identification within the last decade of the full set of autophagy genes in yeast, the discovery of human homologues tied to specific disease states, and the definition of signaling pathways regulating autophagy have accelerated interest in elucidating the full molecular details of this important process.

General Mechanism

Autophagy consists of signaling, sequestration of cytoplasm, completion of vesicle formation, targeting of the completed vesicle to the lysosome/vacuole followed by docking and fusion, and breakdown. In higher eukaryotes, the lysosomal pathway of intracellular degradation is further partitioned into three distinct pathways: macroautophagy, chaperone-mediated autophagy, and microautophagy. Macroautophagy is the subject of this brief review.

Macroautophagy begins with formation in the cytoplasm of the autophagosome, a double membrane vesicular structure. The autophagosome engulfs cytoplasmic proteins, lipids, and damaged organelles such as mitochondria, endoplasmic reticulum, and ribosomes. The autophagosomal outer membrane fuses with the lysosome in mammalian cells to deliver the sequestered cargo. The inner membrane of the fused structure (autophagolysosome), dissolves, and digestion of interior contents by lysosomal hydrolytic enzymes generates nucleotides, amino acids, and free fatty acids that can be recycled to provide raw materials and energy to the cell.

Morphology

Upon autophagy induction, a membrane cisterna known as the isolation membrane appears and curves around part of the cytoplasm. Sealing of the membrane edges results in the double-membraned autophagosome, visible by electron microscopy. Autophagosomes then fuse with a lysosome, where degradation of the delivered material occurs. Isolation and autophagosome membranes differ from other cellular membranes in having few intramembrane proteins.
Autophagic Proteins/Signaling Pathways

In eukaryotic cells, autophagy occurs at constitutive low levels for housekeeping functions such as destruction of dysfunctional organelles. Upregulation occurs in the presence of external stressors (starvation, hormonal imbalance, oxidative stress, extreme temperature, and infection), and internal needs (generation of source materials for architectural remodeling, removal of protein aggregates). Autophagy is regulated by various kinases, phosphatases, and guanosine triphosphatases (GTPases). For example, mediators of phosphoinositide-3 (PI3) kinase signaling pathways and trimeric G proteins play roles in regulating formation of autophagosomes. Target Of Rapamycin (TOR) kinase, a predominant negative regulator of autophagy, is a significant target for cancer therapeutics. The eukaryotic Initiation Factor 2 (eIF2) kinase Gcn2 and its downstream target Gcn4, a transcriptional transactivator of autophagy genes, induce autophagy under conditions of cellular starvation. The PI3K/Akt signaling pathway inhibits autophagy in response to insulin-like and other growth factor signals. Downstream of TOR kinase, a range of autophagy proteins participate as follows:

- Protein serine/threonine kinase complex that relays upstream signals from TOR kinase: Atg1, Atg13, Atg17
- Lipid kinase signaling complex that engages vesicle nucleation: Atg6, Atg14, Vps34, and Vps15
- Ubiquitin-like conjugation pathways that facilitate vesicle expansion: LC3 (Atg8) and Atg12 networks
- Recycling pathway for removal of autophagy proteins from autophagosomes: Atg2, Atg9, Atg18

LC3 (rat microtubule-associated protein light chain 3), localizes in autophagosome membranes after processing, and is a classical autophagy marker. Following LC3 synthesis, Atg4 cleaves the C-terminus to produce cytosolic LC3-I. LC3-I is converted to LC3-II by Atg7 and Atg3. LC3-II is modified by phosphatidylethanolamine (PE) at the C-terminus and binds to autophagosomal membrane. The amount of LC3-II correlates with extent of autophagosome formation.

Beclin1, a Bcl-2 interacting protein, stimulates autophagy when overexpressed in mammalian cells. Beclin1 is monoallelically deleted in human breast and ovarian cancers, with reduced expression in those tumors. Beclin1 overexpression promotes autophagy and inhibits tumorigenesis in breast carcinoma cells; conversely, heterozygous disruption of Beclin1 promotes tumorigenesis in mice. Beclin1 associates with the human class III phosphatidylinositol 3- kinase (PI3K), hVps34. The lipid product of Vps34, PI(3)P, is required for autophagy, and also for assembly of proteins involved in endocytosis and trafficking of enzymes from the trans-Golgi network to the lysosomes. Beclin1 is required for hVps34 to function in autophagy.
Autophagy, Apoptosis, and Cell Death

Interplay between autophagy and apoptosis is complex, with autophagy acting either antagonistic, agonist, or independent of canonical programmed cell death via apoptosis.

Examples where autophagy precedes and may even trigger apoptosis include 1) a dramatic increase in autophagy followed by induction of apoptosis in primary sympathetic neurons deprived of neural growth factor, and 2) a similar effect in TNF-α-induced apoptosis of T-lymphoblastic leukemia cell lines, although in this case additional pro-death factors must be present in concert with autophagy to promote apoptosis.

Autophagy inhibitors delay apoptosis while caspase inhibitors do not impair autophagy, indicating that autophagy may be a precedent to caspase-dependent cell death.

On the other hand, enhanced induction of apoptosis in autophagic deficient HT-29 colon carcinoma cells by sulindac sulfide highlights that autophagy under certain cases blocks the apoptotic pathway. Destruction of damaged mitochondria by autophagy may serve to delay signaling for the apoptotic cascade.

In other cases, cells switch between cell death via autophagy or apoptosis, providing a backup for self-execution if the pathway of first choice is corrupted. Physical interactions between autophagic and apoptotic proteins (e.g., beclin1, BNIP3) suggest an intricate system of cell death regulation not previously appreciated.

References

Autophagy in the Research Spotlight

LC3 is currently the standard marker of autophagosomes. Tracking the conversion of LC3-I to LC3-II, occurring via conjugation to phosphatidylethanolamine, is an indicator of autophagic activity. Due to the transient nature of autophagosomal structures, the ratio of LC3-II to LC3-I can be used to estimate the course of autophagic activity over time. Under autophagic conditions due to external or internal stressors, western blotting of LC3 usually detects two bands: cytosolic LC3-I (18 kDa) and autophagosome associated LC3-II (16 kDa).

LC3 Monoclonal and Polyclonal Antibodies

Abgent LC3 antibodies are specific for the hallmark autophagy protein LC3, which associates with autophagosomal vacuoles (autophagosomes) formed upon induction of autophagy. These antibodies have proven to be a successful autophagy detection tool (Fig. 1). LC3 polyclonal (Cat. #AP1801a) and LC3 monoclonal (Cat. #AM1800a) are validated for use in western blotting, immunoprecipitation and immunofluorescence (Fig. 2).

Autophagy Antibodies with the Right Immunogenic Design

Our expanding autophagy line now includes over 150 novel autophagy antibodies directed against human homologs of a series of proteins identified in the autophagy pathway including polyclonal and monoclonal antibodies against a range of epitopes spanning LC3, a marker for the autophagic process. These antibodies are characterized by immunoblot and cell staining of a range of human and rodent lysates.

Abgent has numerous citations for its LC3 products.

Please visit www.abgent.com for a full listing.

Select Citations:

Autophagy Hallmark Target: LC3

Abgent has developed one of the most validated set of LC3 antibodies available. LC3 is a major marker of the autophagy process, and Abgent’s premium LC3 selection serves as a vital tool for autophagy research.

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1. IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.
2. IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.
3. WB analysis of rat brain tissue lysate using the LC3 antibody.
4. IF analysis of U251 cells treated with Chloroquine using the LC3 antibody.
5. IF analysis of U251 cells using the LC3 antibody.
6. WB analysis of CHO cell line lysates using the LC3 antibody. Native LC3 and LC3 S12 mutant vectors were transfected to show phosphorylation specificity.
## Autophagy Antibodies

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## Notes

1. WB analysis of A2058 cell lysate using the APG1 antibody.
2. IHC analysis of human hepatocarcinoma tissue stained with ATG3 antibody.
3. IF analysis of U251 cells treated with Chloroquine using the ATG4B antibody.
4. WB analysis of Jurkat cell line lysate with the APG4C antibody.
5. IF analysis of U251 cells treated with Chloroquine using the ATG5 antibody.
6. WB analysis of Cos7, HEK293, MEF, and HeLa cell lysates using the ATG7 antibody.
7. WB analysis of mouse heart tissue lysate using the ATG9A antibody.
### Autophagy Antibodies

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#### RAB24 & RGS19

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#### AMBRA, CAMKV, DRAM, GABARAP & PIST

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#### Additional Images:

9. IF analysis of U251 cells treated with Chloroquine using the ATG12 antibody.
10. WB analysis of HeLa cell line lysate with the ATG16L antibody.
11. IHC analysis of human breast carcinoma tissue stained with the Beclin1 antibody.
12. WB analysis of non-transfected and transfected 293 cell lysates using the LAMP2 antibody.
13. IHC analysis of human breast carcinoma tissue stained with the Beclin1 antibody.
14. WB analysis of non-transfected and transfected 293 cell lysates using the LAMP2 antibody.
Autophagy Antibodies (cont.)

**PI3KC3**

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**SQSTM1 (p62) & UVRAG**

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16. WB analysis of HEK-293 cell lysates using the PI3KC3 antibody.
17. IHC analysis of human skeletal muscle tissue stained with the PI3KR1 antibody.
18. WB and IP analysis of 293T cell lysates using the UVRAG antibody.
19. IF analysis of U251 cells treated with Chloroquine using the SQSTM1 (p62) antibody.
Additional Autophagy Products

Blocking Peptides:
Blocking peptides are available for all Abgent autophagy antibodies. Check the technical data sheet of any autophagy antibody at www.abgent.com to locate the companion blocking peptide.

Synthetic Peptides:
Abgent offers a host of synthetic peptides useful in autophagy research. A selection of synthetic peptides are listed here. For a full list, please visit the Abgent website.

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Transfected Cell Lysates:
Transfected cell lysates are available for a number of Abgent autophagy antibodies. Check the technical data sheet of any autophagy antibody at www.abgent.com to locate the companion transfected cell lysate.

AutoDOT:
The new autophagy visualization dye! Superior to traditional monodansyl cadaverine staining.
- Faster penetration
- Higher sensitivity/greater signal endurance on stored slides
- Greater resistance to acid

MDHx staining in cerebellar cells
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